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The relationship of plasma urea nitrogen with growth traits and age at first estrus in gilts^{1,2}

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ABSTRACT: Gilts that reach puberty at an earlier age with more backfat have greater lifetime productivity. Increased growth rates generally promote earlier age at first estrus; however, an association of age at first estrus with discrete measures of body fatness remains controversial. We tested the hypothesis that metabolic state as determined by concentrations of plasma urea nitrogen (PUN), which reflect lean tissue growth, were correlated with age at first estrus. Blood samples were collected from gilts ($n = 337$) at 102, 123, and 145 d of age during development. Concentrations of albumin, creatinine, glucose, and PUN were determined. Body weight and backfat thickness were determined at each time point. From 130 to 240 d of age, gilts were monitored for first pubertal estrus. Concentrations of creatinine increased whereas concentrations of glucose decreased with increasing age ($P < 0.0001$). Concentrations of albumin and PUN remained relatively stable throughout

development. Average daily BW gain ($r = 0.22$) and change in backfat thickness ($r = 0.29$) had a positive phenotypic correlation ($P < 0.0001$) with PUN at 145 d of age. Concentrations of PUN at 102 and 123 d of age were not phenotypically correlated with pubertal age, but there was a moderately negative phenotypic correlation ($r = -0.22$; $P < 0.0001$) of PUN at 145 d of age with age at first estrus along with a negative genetic correlation ($r = -0.42$). The relationship of PUN with age at first estrus shifted from linear to quadratic with advancing age. These data demonstrate that near the age at which gilts are selected for entry into the breeding unit, those with greater PUN have increased BW and backfat thickness and display pubertal estrus earlier but that PUN does not account for additional variation in age at first estrus beyond growth rate or backfat. It is concluded that PUN can be used to select gilts with increased efficiency of nutrient use without negatively impacting pubertal development.

Key words: estrus, gilt, growth, puberty, urea nitrogen

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INTRODUCTION

Puberty is an important determinant of sow lifetime productivity (Patterson et al., 2010). It has long been believed that achieving a critical threshold in BW or composition is necessary for first estrus in gilts (Kirkwood and Aherne, 1985). Increased growth rates generally promote early attainment of puberty (Beltranena et al., 1991), but investigations into the relationship of age at first estrus with discrete measures of body fatness have not yielded consistent results (see Bortolozzo et al., 2009). Moreover, continued selection of maternal-line gilts for increased lean and decreased fat deposition has likely altered these relationships (Gaughan et al., 1995).

Selecting gilts for increased leanness can have detrimental impacts on reproductive performance (Rauw

et al., 1998). Cameron et al. (1999) concluded that selection for greater lean tissue growth rate increased efficiency of nutrient use without negatively impacting reproductive performance of gilts but acknowledged that the threshold for body fat content may be more critical in lean genotypes. More recently, Patterson et al. (2002) reported that when lean tissue growth is not limiting, differences in fatness were not related to puberty. However, this is dependent on where along the growth curve measurements are made. Composition of tissue accretion is related to metabolic state, which may be a more important determinant in the timing of first estrus than discrete measures of fat or protein (Rozeboom et al., 1995).

Dietary AA in excess of requirements for tissue repair and deposition are converted to urea in the liver (Rodwell, 1993). Concentrations of plasma urea nitrogen (PUN) are therefore indicative of lean tissue deposition in growing pigs (Coma et al., 1995). To test the hypothesis that metabolic state during development is related to onset of puberty, we evaluated the relationship of PUN at different time points during gilt development with age at first estrus.

MATERIALS AND METHODS

All animal procedures were reviewed and approved by the U.S. Meat Animal Research Center Animal Care and Use Committee and conducted in accordance with the *Guide for the Care and Use of Agricultural Animals in Research and Teaching* (FASS, 2010).

Animals were from a Yorkshire–Landrace–Duroc resource population (Kuehn et al., 2009; Lindholm-Perry et al., 2009) developed for identification of QTL for production traits. Gilts were from three 3-wk farrowing groups: 133 were born in May, 97 were born in July, and 107 were born in November. Sows were either parity 1 ($n = 170$) or parity 2 ($n = 34$). Gilts were weaned at 15 to 21 d of age and placed in a nursery building until 66 d of age, after which time they were moved to a finishing building in pens of 8 to 12 gilts. At approximately 186 d of age, 108 gilts (36/farrowing group) that had displayed at least 1 estrus were removed from the finishing unit to meet breeding needs of the farm. Animals were allowed ad libitum access to corn–soybean meal diets (Table 1) formulated to meet or exceed requirements (NRC, 1998). For detection of estrus, gilts were allowed daily fence line contact with mature boars (>11 mo of age) as the herdsman applied back pressure to gilts within each pen and observed for signs of estrus. Estrus detection commenced at 130 d of age and continued until 240 d of age. Only gilts that displayed pubertal estrus by 240 d of age were included in the study ($n = 336$).

Blood samples were collected from each animal by jugular venipuncture at 102.3 ± 0.2 , 123.4 ± 0.2 ,

and 145.0 ± 0.2 d of age (mean \pm SE), which spans the portion of the growing–finishing period when most of the feed is consumed during the linear portion of the growth curve, to provide accurate estimates of PUN. Because animals were allowed ad libitum access to food, blood samples were considered to be collected during the fed state (Klindt et al., 2006). Blood samples were collected into 10 mL heparinized syringes and immediately placed on ice. Within 4 h of collection, plasma was obtained by centrifugation ($2,000 \times g$ for 30 min at 4°C), aspirated, and frozen (-20°C) until analysis. Plasma samples were analyzed for albumin, creatinine, glucose, and urea N using a Technicon Autoanalyzer (Marsh et al., 1965). Within 24 h of blood collection, BW and backfat thickness were recorded. Backfat was measured ultrasonically (Lean-Meater; Renco Corp., Minneapolis, MN) 5 to 8 cm lateral to the midline at the first rib and last rib, and backfat measurements were averaged to obtain a single estimate of overall backfat thickness.

Average daily BW gain and average daily change in backfat thickness when the pigs were in the finishing facility were determined by within-pig linear regression using the 3 recorded BW and average BF estimates in a mixed model that included sire within farrowing group as a random effect. To compare the accretion of BW and backfat among animals at a standard age, parameters of the linear regression equations were used to predict BW and backfat at 21 wk of age for each pig. The effect of age on BW, backfat thickness, and concentrations of metabolites in plasma were determined using mixed model ANOVA fitting sire, farrowing group, and litter as random effects. Denominator degrees of freedom were calculated using the Kenward-Roger approximation. Phenotypic correlation coefficients for age at first estrus with concentrations of metabolites in plasma were generated with the PROC CORR statement (SAS Inst. Inc., Cary, NC). The relationship of PUN with age at first estrus was determined using a mixed model that included sire and farrowing group and litter as random effects. Partial correlations of age at first estrus with PUN were obtained using the MANOVA/printe option in the GLM statement of SAS after accounting for either backfat or lifetime growth rate. Lifetime growth rate was defined as BW minus birth weight divided by age in days. To estimate the genetic correlation of PUN with age at first estrus, 2-trait models were fit using MTDFREML (Boldman et al., 1995). The effects of lifetime growth rate and body fatness on PUN and age at first estrus were determined with a mixed model ANOVA that included sire and farrowing group and litter as random effects.

Table 1. Diets used during the course of the study

Item	Grower 1 ¹	Grower 2 ²	Finisher ³	Breeding ⁴
Ingredient, % as fed				
Ground corn	70.65	76.28	79.08	80.66
Soybean meal, 44% CP	25.01	20.02	17.55	10.81
Alfalfa meal, dehydrated	—	—	—	4.00
Soybean oil	1.00	1.00	1.00	1.00
Dicalcium phosphate	1.17	0.76	0.61	1.6
Ground limestone	0.96	0.84	0.82	0.70
Sodium chloride	0.30	0.30	0.30	0.50
Vitamin mix ⁵	0.20	0.20	0.20	0.20
Trace mineral mix ⁶	0.20	0.20	0.20	0.20
L-Lys	0.24	0.17	0.12	—
Thr	0.06	0.02	0.01	—
Met	0.01	—	—	—
Choline chloride	0.10	—	—	0.20
Chlortetracycline 50	0.10	0.10	—	—
Tylan 40	—	—	—	0.125
BMD ⁷	—	—	0.03	—
Total	100.00	100.00	100.00	100.00
Calculated nutrient composition, DM basis				
ME, kcal/kg	3,353	3,377	3,388	3,274
CP, %	18.00	16.00	15.00	12.60
Calcium, %	0.75	0.60	0.55	0.72
Phosphorus, %	0.59	0.49	0.46	0.61
Digestible Lys, %	1.18	0.83	0.73	0.47

¹Diet was fed from 56 to 90 d of age.²Diet was fed from 90 to 112 d of age.³Diet was fed from 112 d of age until gilts were moved to breeding at 160 d of age.⁴Daily amount fed per gilt was 2.72 kg.⁵Vitamin A (retinyl acetate), 2,200,000 IU/kg; vitamin D₃ (cholecalciferol), 440,000 IU/kg; vitamin E (DL- α -tocopheryl acetate), 17,600 IU/kg; vitamin K (menadione sodium bisulfate complex), 2,200 mg/kg; niacin, 22,000 mg/kg; D-pantothenic acid (D-calcium-pantothenate), 12,100 mg/kg; riboflavin, 4,400 mg/kg; and vitamin B₁₂, 22 mg/kg.⁶Ferrous sulfate heptahydrate, 35.05%; copper sulfate pentahydrate, 1.77%; manganese oxide, 9.62%; calcium iodate, 0.016%; sodium selenite, 0.033%; and calcium carbonate, 50.91%.⁷BMD = bacitracin methylene disalicylate.

RESULTS

Means for age at first estrus and blood metabolites for gilts are listed in Table 2. Body weight and backfat thickness increased ($P < 0.0001$) linearly during the developmental grow–finish period (Table 3). Concentrations of albumin in plasma did not differ with age (Table 3). Concentrations of creatinine in plasma were least ($P < 0.0001$) at 102 d of age and similar at 123 and 145 d of age (Table 3). Glucose in plasma decreased ($P < 0.0001$) linearly through development (Table 3). Concentrations of PUN were less ($P < 0.04$) at 123 d of age than at 102 and 145 d of age, which did not differ from each other (Table 3). Average daily BW gain and change in backfat of gilts were near expected ranges and are listed in Table 4.

Phenotypic correlations for PUN and growth traits are reported in Table 5. Average daily gain had a strong

Table 2. Descriptive statistics for age at first estrus and metabolites in plasma of gilts

Variable ¹	Age, d	<i>n</i>	Mean	SD	Minimum	Maximum
Pubertal estrus, d	—	336	181.4	14.8	139	222
ALBU	102	336	33.9	3.8	20.2	44.7
	123	335	34.3	3.8	20.1	44.6
	145	334	34.5	3.9	15.4	45.9
CREAT	102	337	1.11	0.16	0.63	1.91
	123	336	1.21	0.16	0.82	1.68
	145	336	1.28	0.18	0.85	1.78
GLUC	102	337	26.5	7.2	7.4	66.5
	123	336	24.5	7.1	6.6	58.8
	145	336	23.0	6.9	7.3	57.2
PUN	102	337	7.01	2.54	3.31	33.26
	123	336	6.68	2.08	2.79	21.63
	145	336	7.08	2.03	2.81	20.26

¹ALBU = albumin, mg/mL; CREAT = creatinine, mg/dL; GLUC = glucose, mg/mL; PUN = plasma urea nitrogen, mg/dL.

positive correlation ($P < 0.0001$) with average daily change in backfat as well as BW and backfat at 21 wk of age (Table 5). Concentrations of PUN at different time points were positively correlated ($P < 0.0001$) with each other (Table 5). The strength and magnitude of these correlations decreased as time between measures increased. Average daily BW gain was correlated ($P < 0.0001$) with PUN at 145 d only (Table 5) whereas average daily change in backfat was positively correlated ($P < 0.0001$) with PUN at both 123 and 145 d of age (Table 5). Body weight at 21 wk was correlated with PUN at 123 d ($P < 0.01$) and 145 d ($P < 0.0001$) of age whereas backfat thickness at 21 wk was positively correlated ($P < 0.001$) with PUN at all time points (Table 5).

Age at first estrus was not phenotypically correlated with any of the blood metabolites measured at 102 and 123 d of age (Table 6). Neither creatinine nor glucose at 145 d of age was correlated with pubertal age. At 145 d of age, concentrations of both albumin and PUN had a negative phenotypic correlation with age at first estrus (Table 6), but the correlation was more significant for PUN ($P < 0.0001$) than for albumin ($P < 0.001$). Genetic correlations for age at first estrus with PUN at 102, 123, and 145 d of age were -0.09 , -0.05 , and -0.42 , respectively. The regressions of PUN with age at first estrus are listed in Table 7. The residual phenotypic correlations for age at first estrus with PUN at 145 d of age were -0.07 ($P = 0.36$) and -0.10 ($P = 0.20$) after adjusting for lifetime growth rate and backfat thickness, respectively. Gilts were stratified base on the lifetime growth rate threshold of 600 g/d and their relative amounts of adipose tissue accretion. Data in Table 8 confirm that fatter gilts have greater PUN and gilts with greater growth rate reached puberty earlier. Correlations for BW and ADG at 21 wk of age with age at first estrus were linear ($r = -0.50$ and -0.52 , respectively; $P < 0.0001$).

Table 3. Least squares means for BW, backfat thickness, and metabolites in plasma collected from gilts during development at 102, 123, and 145 d of age

Variable ¹	Age class, d			SEM	P-value
	102	123	145		
BW, kg	51.3 ^a	67.9 ^b	85.4 ^c	0.6	0.0001
Backfat, mm	10.45 ^a	12.47 ^b	14.58 ^c	0.2	0.0001
Albumin, mg/mL	33.7	34.2	34.4	0.4	0.1122
Creatinine, mg/dL	1.12 ^a	1.22 ^b	1.29 ^c	0.02	0.0001
Glucose, mg/mL	26.3 ^a	24.3 ^b	22.8 ^c	0.7	0.0001
PUN, ² mg/dL	7.00 ^a	6.67 ^b	7.07 ^a	0.17	0.0316

^{a-c}Means within a row without a common superscript are different ($P < 0.05$).

¹Random source of variation: sire within farrowing group.

²PUN = plasma urea nitrogen.

DISCUSSION

Age at first estrus is influenced by physiological maturity of the gilt, which is related to the composition of BW gain during gilt development (Kirkwood and Aherne, 1985). Concentrations of PUN are an easily measured index of efficiency of AA use for lean tissue growth (Coma et al., 1995). Dietary AA in excess of those required for tissue repair and accretion are not stored but rather are deaminated and the ammonia converted to urea in the liver. Differences in lean tissue deposition are therefore reflected in PUN concentrations, which are heritable (Klindt et al., 2006) and can be used to select pigs with greater potential for lean growth. Rozeboom et al. (1995) speculated that attainment of puberty was related to a metabolic state at a critical period in development. Our hypothesis was that concentrations of PUN during development reflected differences in metabolic state and would be associated with differences in age at puberty. In support of this, we observed negative phenotypic and genetic correlations for age at first estrus with concentrations of PUN at 145 d of age in developing gilts.

It might be possible to include measures for blood markers in a multivariate model to account for the influence

Table 5. Pearson correlation coefficients for plasma urea nitrogen (PUN) measured at 102 (PUN102), 123 (PUN123), or 145 (PUN145) d of age with growth traits during development in gilts

Variable ¹	PUN102	PUN123	PUN145	ADG	BW21WK	ADCBF
PUN123	0.60 [‡]					
PUN145	0.49 [‡]	0.80 [‡]				
ADG	-0.07	0.09	0.22 [‡]			
BW21WK	0.09	0.17 ^{**}	0.25 [‡]	0.76 [‡]		
ADCBF	0.03	0.22 [‡]	0.29 [‡]	0.51 [‡]	0.34 [‡]	
BF21WK	0.20 [†]	0.34 [‡]	0.34 [‡]	0.43 [‡]	0.52 [‡]	0.76 [‡]

¹ADG = average daily BW gain from approximately 102 to 145 d of age; BW21WK = predicted BW at 21 wk of age; ADCBF = average daily change in backfat thickness from approximately 102 to 145 d of age; BF21WK = predicted average backfat thickness at 21 wk of age ($n = 337$ to 336).

^{**} $P < 0.01$; [†] $P < 0.001$; [‡] $P < 0.0001$.

Table 4. Descriptive statistics for calculated variables

Variable	<i>n</i>	Mean	SD	Minimum	Maximum
ADG, ¹ kg/d	337	0.81	0.13	0.38	1.41
BW21WK, ² kg	337	85.3	9.3	51.9	114.4
ADCBF, ³ mm/d	337	0.10	0.05	-0.04	0.31
BF21WK, ⁴ mm	337	14.72	2.69	7.75	24.17
LGR21, ⁵ g/d	337	580	62.1	345.5	748.3

¹ADG = average daily BW gain from approximately 102 to 145 d of age.

²BW21WK = predicted BW at 21 wk of age.

³ADCBF = average daily change in backfat thickness from approximately 102 to 145 d of age.

⁴BF21WK = predicted average backfat thickness at 21 wk of age.

⁵LGR21 = lifetime growth rate from birth to 21 wk of age.

of metabolic state on pubertal development when selecting gilts. Leptin seems an obvious choice because it is reflective of adipose tissue mass and stimulates the reproductive neuroendocrine axis of the gilt (Barb et al., 2005). There was a strong negative genetic correlation (-0.63) between age at first estrus and concentrations of leptin at puberty in this resource population, but leptin measured at 128, 149, and 180 d of age was not phenotypically correlated with age at puberty ($r < 0.05$; Kuehn et al., 2009). Moreover, Patterson et al. (2002) reported that concentrations of leptin in gilts at 135 d of age had no phenotypic relationship with age at puberty when lean tissue growth was not limiting. The current observation of negative phenotypic and genetic correlations of PUN measured at 145 d of age with age at first estrus is likely a consequence of the fact that PUN is reflective of the relative rates of protein and adipose tissue accretion during this period of development whereas leptin reflects fat mass alone. It is important to note, however, that concentrations of PUN can also be affected by feeding excess AA or protein. This illustrates the importance of formulating diets to meet protein and AA requirements for growth.

In this study, gilts with greater PUN had greater 21 wk BW and backfat thickness. The phenotypic correlations of PUN with these growth traits as well as ADG and daily change in backfat thickness were similar in magnitude to those of the earlier generations of this resource population reported previously by Klindt et al. (2006) and reinforces the supposition that gilts with greater growth rates generally reach puberty at younger ages (Beltranena et

Table 6. Pearson correlation coefficients for age at first estrus with plasma concentrations of albumin (ALBU), creatinine (CREAT), glucose (GLUC), and plasma urea nitrogen (PUN) measured at 102, 123, and 145 d of age in gilts during development ($n = 336$)

Age, d	ALBU	CREAT	GLUC	PUN
102	-0.01	0.07	-0.09	-0.04
123	-0.10	0.04	-0.11	-0.08
145	-0.18 [†]	0.06	-0.11	-0.22 [‡]

[†] $P < 0.001$; [‡] $P < 0.0001$.

Table 7. Relationship of plasma urea nitrogen (PUN) measured at 102, 123, and 145 d of age during development with age at first estrus

Variable	Age, d	Regression estimates ¹ (± SE)					
		Intercept	P-value	Linear	P-value	Quadratic	P-value
PUN	102	185.83 (±5.31)	0.0001	-0.69 (±0.80)	0.3901	0.01 (±0.03)	0.6385
	123	192.63 (±6.25)	0.0001	-2.19 (±1.22)	0.0746	0.07 (±0.06)	0.2389
	145	204.43 (±6.60)	0.0001	-4.28 (±1.25)	0.0007	0.14 (±0.06)	0.0297

¹Random sources of variation: sire and farrowing group.

al., 1991; Kummer et al., 2009; Tummaruk et al., 2009). It is generally accepted that in the pig, growth rate and associated metabolic traits limit age at first estrus up to a certain threshold point. The relationship of PUN with age at first estrus shifts from linear at younger ages to quadratic at 145 d of age indicating a curvilinear pattern of growth and suggesting that above a certain point, increasing concentrations of PUN have little impact on the timing of pubertal attainment.

Klindt et al. (1999) reported that small but significant differences in BW and backfat thickness at the start of boar exposure affected the rate at which gilts obtained puberty. Gilts in our study weighed approximately 85 kg at 21 wk and PUN was phenotypically correlated with 21 wk backfat thickness. When adjusted for backfat thickness or lifetime growth rate from birth to 145 d of age, the residual correlation of PUN with age at first estrus was not significant. Therefore, PUN is not accounting for additional variation in age at first estrus. Direct determination of individual daily BW gain or backfat is not always possible as many farms lack the facilities, equipment, and expertise to accomplish this. In such cases, PUN could serve as an effective and economically viable alternative trait.

Faster growing gilts typically reach puberty earlier. Some believe that there is a critical threshold in lifetime daily BW gain that determines when the rate of sexual development is limited by growth (Kummer et al., 2009; Rozeboom et al., 1995). It is generally accepted that this threshold is close to 600 g/d (Beltranena et al., 1991). To more fully explore the relationships between PUN and age at first estrus and to gain a greater understanding of PUN as a measure of gilt body tissue development, gilts were grouped into 4 discrete classes based on the suggested threshold for daily gain from birth to selection and accretion of adipose tissue. Gilts in this study had an average lifetime growth rate of 580 g/d from birth to 145 d of age with 38% having a daily BW gain above the proposed 600 g/d threshold. The overall positive relationship of adiposity and PUN were clearly evident. Body weight and age at first estrus were not different between greater growth gilts that were depositing more fat compared with the 30% (37 of 128) of the greater growth gilts that were depositing less

Table 8. Plasma urea nitrogen (PUN) concentrations and age at first estrus in gilts with differing combinations of lifetime growth rate (LGR) and backfat thickness at 21 wk of age

Variable	Group ¹				Pooled SE
	HGHF	HGLF	LGHF	LGLF	
No.	91	37	73	135	—
BW, kg	95.0	93.8	83.8	78.3	1.1
LGR21 ² , g/d	644 ^a	636 ^a	563 ^b	531 ^c	5
Backfat, mm	17.33 ^a	13.04 ^b	16.16 ^c	12.58 ^b	0.25
PUN21 ³ , mg/dL	8.08 ^a	6.63 ^b	7.68 ^a	6.37 ^b	0.35
Age at first estrus, d	174.1 ^a	174.6 ^a	182.6 ^b	188.0 ^c	3.1

^{a-c}P < 0.01.

¹HGHF = greater growth, greater fat; HGLF = greater growth, lower fat; LGHF = lower growth, greater fat; LGLF = lower growth, lower fat.

fat and had reduced concentrations of PUN. The greater-growth greater-fat gilts probably had less lean tissue mass potential compared with the faster growing leaner gilts, which may not have been as far along their respective growth curve. Gaughan et al. (1997) found that gilts that were leaner at 140 d of age deposited more fat from selection to puberty than gilts that were fatter at selection. These data indicate that the relationship of age at first estrus with the suggested threshold in lifetime growth rate is more dependent on total BW gain than the composition of growth. Age at first estrus was intermediate for the slower growing gilts that had deposited more fat when compared with faster-growing gilts or slower-growing leaner gilts. The slower-growing fatter gilts may have had decreased genetic potential for lean tissue growth or may have been impacted by prenatal factors (e.g., low birth weight gilt) that altered their developmental potential for muscle growth. Nonetheless, all the gilts in this study reached puberty at commercially acceptable ages.

Selecting gilts based on traditional standards of efficiency (i.e., feed conversion ratio) will lead to increased mature size resulting in females that are later maturing (Kirkwood and Aherne, 1985) and selection for leanness can decrease reproductive performance (see Rauw et al., 1998). Cameron et al. (1999) concluded that selection for greater efficiency of lean tissue growth rate increased efficiency of nutrient use without negatively impacting reproductive performance of gilts. The heritability of PUN measured at 107, 128, and 149 d of age was 0.35, 0.21, and 0.19, respectively (Klindt et al., 2006). Thus, PUN is an easily measured, heritable trait that could be used to select animals with greater efficiency of AA use for accretion of lean tissue. Klindt et al. (2006) proposed that selecting for reduced concentrations of PUN (i.e., efficient use of dietary AA for lean growth) would result in beneficial outcomes in amount of lean tissue accretion, carcass fatness, and decreased N excretion and concentration in manure. The positive results of this for increasing efficiency of producing marketable pork and reducing the impact of production on

the environment are obvious; however, the consequences of such a selection strategy on gilt development and sow productivity were not considered. The current data indicates that if selection pressure was applied to PUN, especially at early ages in the growth phase, the positive benefits of increased nutrient use and lean tissue growth could be achieved without negatively impacting pubertal development of gilts. The positive phenotypic association of PUN measured at 102 d with backfat thickness at 21 wk was mirrored by a positive genetic correlation ($r = 0.65$) that tended ($P < 0.1$) toward significance and confirms the previous observation by Klindt et al. (2006). Given the importance of BW and backfat thickness on productivity and longevity of sows (Bortolozzo et al., 2009), selection against concentrations of PUN in development may have implications for sow productivity, which merits further consideration.

In conclusion, concentrations of PUN are heritable and correlated with BW and backfat thickness in gilts. The main finding of this study is that the use of PUN to select gilts with increased efficiency of nutrient use would not be expected to negatively impact pubertal development; however, the impact that such a selection strategy would have on lifetime productivity of gilts requires further investigation.

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